## Clean Copy of Claims

18. (Currently amended) A method for inhibiting or reversing metastasis in a M+ class tumor, wherein said tumor is capable of existing in M+ and MO classes, comprising the step of contacting said with an effective amount and for an effective period of time with an inhibitor of the upregulation (overexpression) of a gene identified as being associated with said M+ class, said gene identification being made by a genetic method comprising the steps of:

A. Identifying by expression-profiling of tumor sample cohorts of said M+ and MO classes of sald tumor, coupled with permutational statistical analysis, to generate a candidate gene list, those genes whose expression differ statistically between said classes of said tumor and that are upregulated in the M+ class and downregulated in the MO class;

B. producing a class-predictive algorithm based upon said predictive genes with a permutational *P* value of <0.05; and,

C. applying said algorithm to a candidate tumor to produce a Predictive Strength value that will assign the M+ or MO class to said tumor, wherein said algorithm comprises two primary equations:

(1) 
$$v_i = [x_i - (\mu_{Mo} + \mu_{M+})/2]$$

wherein vi is the selective vote, xi is the expression level in the tumor sample, and  $\mu$ MO and  $\mu$ M+ are the metastatic classes of reference samples, and wherein said votes are summed in order to obtain total votes for the non-metastatic ( $V_{Mo}$ ) and metastatic ( $V_{M+}$ ) classes; and,

(2) Prediction Strength = [ 
$$(V_{Mo} - V_{M+}) / (V_{Mo} + V_{M+})$$
 ]

wherein Prediction Strength values range between 0 and 1.

- 19.(Currently amended) The method according to claim 26, wherein said inhibitor is a neutralizing antibody directed against the protein encoded by said upregulated M+ gene.
- 20.(Currently amended) The method according to claim 26, wherein said inhibitor is a chemical inhibitor.
- 21. (Original) The method according to claim 20, wherein said inhibitor is directed against a member of the the metastatic overexpressed gene group consisting of the signal transduction inhibitor STI-571, the RAS inhibitor R115777, the MAP2K1/MAP2K2 protein kinase inhibitor U0126, the specific signal transduction inhibitor of PDGFRA STI-571, the phosphoinositide 3-kinase inhibitor wortannin, the VEGF inhibitor NM3, the MAP kinase inhibitor CC1-779, and the glutathione S-

transferase inhibitor TLK 886.

- 22. (Original) The method according to claim 21, wherein said inhibitor is the RAS inhibitor R115777.
- 23. (Original) The method according to claim 21, wherein said inhibitor is SCH88336.
- 24. (Original) The method according to claim 21, wherein said inhibitor is U0126.
- 25. (Original) The method according to claim 21, wherein said inhibitor is STI-571.
- 26 (New) The method of claim 18, wherein said upregulated tumor gene is the gene for PDGFRA or a gene downstream from said PDGFRA gene.
- 27 (New) The method of claim 26, wherein said downstream gene is selected from the group consisting of RAS, MAP2K1/MAP2K2, phosphoinositide-3-kinase, VEGF, MAP kinase, and glutathione-S-transferase.